REMARKS

Favorable consideration and allowance are respectfully requested for claims 1-5 in view of the foregoing amendments and the following remarks.

Page 1 of the specification is amended to replace the heading "BACKGROUND OF THE INVENTION" with "FIELD OF THE INVENTION". This amendment provides proper syntax for this section heading. No new matter is introduced by this amendment.

The rejections of claims 1-5 under 35 U.S.C. § 103 as obvious over either Lepran et al. or WO 97/46241 (the '241 application) are respectfully traversed.

Claim 1, as amended, recites that the condition being treated is myocardial damage secondary to myocardial infarction in a patient who has suffered a myocardial infarction. Support for this amendment may be found throughout the specification, for instance on page 1, in the section now titled "FIELD OF THE INVENTION" or in the last sentence on page 2. No new matter is introduced by this amendment.

The Office Action indicates that the previously-presented claim 1 was not limited to a method of treating tissue damage after myocardial infarction. As amended, claim 1 is directed to a "method of treating myocardial damage secondary to myocardial infarction." Accordingly, the claim is clearly limited to methods of treating myocardial damage after myocardial infarction. Further, the language "in a patient who has suffered a myocardial infarction" limits the claim to those treatments that are administered after the myocardial infarction.

Lepran et al. discloses a moxonidine *pre-treatment* for arrhythmias induced by, for instance, myocardial ischemia. The author's ultimate conclusion is that moxonidine reduces the incidence of arrhythmias *during* the acute phase of experimental myocardial infarction, see page S14 of Lepran. In order to be present *during* myocardial infarction, the moxonidine must be administered *before* the myocardial infarction. Any treatment effective *during* the acute phase of an evolving myocardial infarction must be administered *before* that myocardial infarction and is

effective because of its operation *during* that myocardial infarction. Lepran provides no discussion or teaching that moxonidine might be of any benefit if administered after myocardial infarction. Thus, according to Lepran et al., the moxonidine is administered *before* myocardial ischemia. In contrast, amended claim 1 relates to a moxonidine treatment of myocardial damage *after* occurrence of a myocardial infarction. Thus, the timing of the treatment is different.

Moreover, while the arrhythmias discussed in the Lepran et al. article may represent abnormal heart operation, they are not indicative of myocardial damage. Myocardial damage does not necessarily lead to arrhythmias. An arrhythmia is not the same as tissue damage. An arrhythmia is a cardiac dysfunction where the heart muscle is not contracting with its normal rhythm. This dysfunctional condition does not amount to and is not at all the same as structural damage of the heart tissue, as is claimed. This is significant because one of skill in the art would understand that the reason for the treatment in Lepran is different from the claimed invention (arrhythmia in Lepran vs. myocardial damage in the claims). Accordingly, Lepran's treatment to correct an irregular heart rhythm is of no relevance to treatments to avoid tissue damage following myocardial infarction.

Thus, the Lepran et al. article does not teach or suggest a method of treating *myocardial damage secondary to myocardial infarction* by administering moxonidine *after* the occurrence of a myocardial infarction. Lepran is different from the claims because Lepran teaches a pre-treatment. Further, that pre-treatment is not provided to treat myocardial damage secondary to myocardial infarction.

The '241 application likewise fails to teach or suggest a method of treatment as is presently claimed. The '241 application relates to the use of moxonidine to treat congestive heart failure. In particular, the '241 application teaches that moxonidine reduces systemic vascular resistance while increasing cardiac output in hypertensive patients (see page 10, lines 24 to 28). The alleged effect of moxonidine in patients suffering from symptomatic congestive heart failure is thus only caused by hemodynamic changes, and not any useful action on damaged myocardial tissue.

These teachings of the '241 application are entirely unrelated to, and have no bearing upon inhibiting tissue damage following myocardial infarction.

Considering the previously-submitted declaration of Professor Dr. Rupp, which concludes that:

> [b]ecause the '241 application deals only with a treatment of congestive heart failure, and because treatments for congestive heart failure are only targeted at improving heart function, and cannot be assumed to be beneficial to inhibit damage secondary to myocardial infarction, the '241 application provides no teaching or suggestion to administer moxonidine to inhibit damage secondary to myocardial infarction.

the teachings of the '241 application are irrelevant to the present application. Thus, this reference fails to teach or suggest the presently claimed method of treating myocardial damage secondary to myocardial infarction in a patient who has suffered a myocardial infarction.

The cited references fail to teach each and every element of the claimed invention. Further, one of skill in the art would have no motivation to try to modify the teachings of the references so as to arrive at the presently-claimed invention.

Accordingly, reconsideration and withdrawal of the rejections under 35 U.S.C. § 103(a) over Lepran and the '241 application are respectfully requested.

CONCLUSION

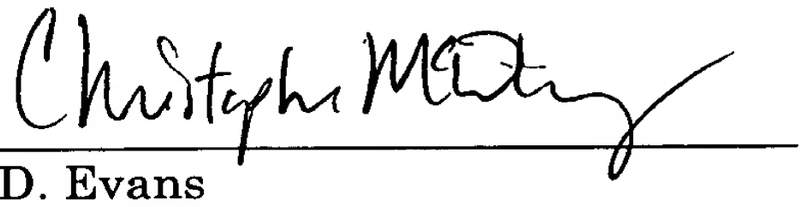
In view of the foregoing, the application is respectfully submitted to be in condition for allowance, and prompt favorable action thereon is earnestly solicited.

If there are any questions regarding this amendment or the application in general, a telephone call to the undersigned would be appreciated since this should expedite the prosecution of the application for all concerned.

If necessary to effect a timely response, this paper should be considered as a petition for an Extension of Time sufficient to effect a timely response, and please charge any deficiency in fees or credit any overpayments to Deposit Account No. 05-1323 (Docket #029300.50194US).

April 19, 2005

Respectfully submitted,



J. D. Evans
Registration No. 26,269

Christopher T. McWhinney
Registration No. 42,875

CROWELL & MORING LLP
Intellectual Property Group
P.O. Box 14300
Washington, DC 20044-4300
Telephone No.: (202) 624-2500
Facsimile No.: (202) 628-8844
JDE:CTM:tlm (371552)